REMARKS

The examiner states that the application contains four inventions or groups of inventions which are not so linked as to form a single general inventive concept under PCT Rule 13.1.

Applicants elect without traverse Group IV, comprising claims 40-42, drawn to a method of monitoring the efficacy of a treatment of a subject having schizophrenia, bipolar disorder, and/or ADHD, comprising comparing the level of expression of at least one gene selected from Tables 1 and 7 in a pre-administration sample and a post-administration sample.

The examiner has also required an election of one species of the generic invention, which species for the elected Group IV are at least one gene selected from Tables 1 and 7 or Table 2, i.e., one combination of gene(s) to which the claims shall be restricted if no generic claim is finally held to be allowable. It is understood however that, upon allowance of a generic claim, applicant will be entitled to consideration of claims to additional species which are written in dependent form or otherwise include all the limitations of an allowed generic claim as provided by 37 CFR 1.141.

For the single species of at least one gene (combination of gene(s)), applicants elect the combination below:

GluR-A (M36418)
NMDA R1 (L08228Exon#22)
NMDA R2 (AF001423)

Glu-binding subunit (Grina) (S61973)

Densin-180 (U66707)

Begain (AF064868)

CAMKII gamma (J04063)

CAMKII beta (M16112)

CAMKII inhibitor alpha (AA85862)

Synapsin II (AI145494)

SNAP-25A (AB003991)

SNAP-25B (AB003992)

VAMP2 (AI101103)

Adenylyl cyclase 2 (AI145367)

Catechol O-methyltransferase (M93257)

Claim 41 is readable on the elected species of a combination of genes with claim 40 being generic.

Schizophrenia is a highly complex neuropsychiatric illness. There is no one gene that predicts the susceptibility to the illness but rather a combination of genes. Moreover, linkage hotspots for schizophrenia are scattered on almost every human chromosome. There is however strong evidence suggesting that aberrant synaptic transmission in the brain is a hallmark feature of schizophrenia. The precise defect in transmission has not yet been identified but the literature supports a profound compromise in excitatory glutamatergic transmission as being integral to the illness. In the prenatal stress animal preparation, several important genes linked to this excitatory neurotransmission are altered.

Favorable consideration and early allowance are respectfully solicited.

Respectfully submitted,

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